1016-38 Haemostatic, Fibrinolytic and Lipid Parameters in Relation to the Initial Manifestation of Coronary Artery Disease

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In order to determine whether haemostatic and fibrinolytic factors relate to the initial manifestation of coronary artery disease (CAD), we measured plasma factor VIIa, fibrin monomer, prothrombin fragment F1 + 2, fibrinogen, antithrombin III, tissue plasminogen activator (t-PA) antigen, t-PA activity, plasminogen activator inhibitor-1 (PAI-1) antigen, plasmin-antiplasmin complex, total serum cholesterol, triglycerides and lipoprotein(a) levels in 42 stable patients with CAD (mean age 63 ± 7.4, range 50–86 years; 32 males). There were 9 hypertensives, 4 diabetics, 15 smokers and 17 with a family history of CAD. Angiographically significant coronary disease was present in 33 patients. The initial manifestation was angina in 26 and MI in 16 patients. A resting, fasting venous blood sample was obtained without venostasis in the morning hours.

On univariate analysis, patients initially manifesting with MI had higher VIIa levels. There was also a trend towards higher fibrin monomer, total cholesterol and triglyceride levels in these patients. Multiple logistic regression produced a model containing factor VIIa, PAI-1 antigen, F1 + 2 and fibrin monomer as having significant association with the mode of presentation (overall $p = 0.005$; pseudo $R^2 = 0.31$):

Variables	Odds Ratio	95% CI	p value
Fibrin monomer	1.42	1.04–1.94	0.025
F1 + 2	1.07	1.00–1.15	0.049
Factor VIIa	14.02	0.81–244	0.07
PAI-1 antigen	0.18	0.04–0.82	0.027

The data indicate an important role of haemostatic and fibrinolytic factors in determining the initial manifestation of CAD. The negative association with PAI-1 antigen may appear contradictory to earlier reports; however, correction for other haemostatic parameters was not always performed in previous studies.

1016-39 Detection of Culprit Territories in the I-123-Metaiodobenzylguanidine (MIBG) Imaging in Cases With Angina Pectoris

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I-123-Metaiodobenzylguanidine (MIBG) is taken up by sympathetic nerve terminals and provides myocardial sympathetic neuronal images. The aim of this study is to evaluate whether I-123-MIBG imaging is useful to indicate ischemic lesion of myocardium. I-123-MIBG images were applied for consecutive 20 patients who were admitted into our hospital with angina pectoris. Thirteen cases were categorized into unstable angina according to the criteria of AHA and other 7 cases were into stable angina. Coronary angiogram was performed simultaneously in all cases. Culprit coronary arteries were indicated with angiographical findings interpreted by uninterested angiographers and other clinical findings. It has been confirmed that the vessels did not cause prior myocardial infarction.

Ischemic territories were detected exactly in 12 of 13 cases (92%) with unstable angina and in 6 of 7 cases (86%) with stable angina with I-123-MIBG imaging. Lesions in LAD were detected in 11 of 13 cases and all 7 lesions in RCA and LCX were detected.

In conclusion, I-123-MIBG imaging is a new useful diagnostic procedure to detect angina related territory and may make it possible to identify culprit coronary lesions in cases with unstable and stable angina pectoris.

1016-40 Blockade of K_{ATP} Channels Does Not Abolish Preconditioning During Demand Ischemia in Man

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Background: Patients with coronary artery disease (CAD) have reduced signs and symptoms of myocardial ischemia during a second stress following an initial ischemic episode, a phenomenon that may reflect preconditioning (PC). K_{ATP} channels have been implicated as a mechanism of PC during no-flow ischemia in animals and man (e.g. angioplasty), but have not been evaluated during demand ischemia. *Methods:* To test the hypothesis that blocking K_{ATP} channels would lessen PC in demand ischemia, 14 pts with CAD were given glibenclamide 10 mg (a dose previously shown to abolish PC during no-flow ischemia) or placebo in a randomized, double-blind fashion prior to either serial exercise tests (ETT) or atrial pacing. The effect of glibenclamide was measured by the reduction in ST depression and/or angina on the second stress. *Results:* Of the 5 pts with serial ETT's, all pts preconditioned, reducing their ST depression on the 2nd ETT an average of 1.46 ± 0.39 mm (SE). This reduction in ST depression was not altered by glibenclamide (1.28 ± 0.34 mm) in cross-over ETT's. PC was also seen with atrial pacing, with the 2nd pacing episode reducing ST depression at maximal heart rate by 0.87 ± 0.05 mm, an effect again not altered by glibenclamide (0.75 ± 0.21 mm). The onset of angina was delayed in the 2nd ETT or pacing stress, and was not altered by glibenclamide. *Conclusions:* Blockade of K_{ATP} channels prior to demand ischemia does not alter the preconditioning effect seen with exercise or atrial pacing. This effect appears to contrast with the role of K_{ATP} channels in no-flow ischemia and suggests that opening of K_{ATP} channels is not a mechanism of PC during demand ischemia in pts with CAD.

1016-41 Presence of Left Ventricular Apical Thrombus Predicts Lack of Myocardial Viability: A Dobutamine Stress Echocardiography Study

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The relationship between the presence of LV apical thrombus and myocardial viability (MV) in pts with coronary artery disease (CAD) has not been previously reported. We therefore studied 80 pts (mean age 65; range 28–85; 64 males, 16 females) with multivessel CAD and LV dysfunction using dobutamine stress echocardiography. Pts were divided into two groups based on echocardiographic visualization of LV apical thrombus: Group 1-definite thrombus in 24 pts (30%) and probable thrombus in 24 pts (30%) versus Group 2-absent in 32 pts (40%).

	Group 1 (n = 48)	Group 2 (n = 32)
Wall Motion Score		
Composite	54.0 ± 5.8*	46.3 ± 6.4
Apical Segments	16.0 ± 2.7*	12.4 ± 3.4
MV Segments/Pt(n)	6.8 ± 4.5	8.1 ± 4.5
Ischemia Segments/Pt(n)	3.3 ± 3.0	3.4 ± 3.0
MV Apical Segments/Pt(n)	0.7 ± 1.2*	1.8 ± 1.3
Ischemia Apical Segments/Pt(n)	0.5 ± 0.9	0.8 ± 1.0

*p = 0.05 versus Group 2

Conclusions: These data suggest that the presence of LV apical thrombus by echocardiography may predict the lack of MV. Thus, baseline echocardiography may identify LV apical segments without MV and obviate need for further MV assessment.

1016-42 Variability of Interpretation for Noninvasive Cardiac Stress Testing: Perfusion Imaging and Echocardiography

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In order to examine the variation of interpretation of noninvasive testing for evaluation of CAD, we examined 32 pts who underwent stress testing in conjunction with both myocardial perfusion imaging (MPI) and echocardiography (E). The studies were performed on separate days, within 6 months of one another. Images were interpreted on two separate occasions by two observers blinded to the clinical information, previous readings, and alternate imaging data. Each reader scored the study segmentally (E = 16, MPI = 20) and for 5 regions. Overall diagnostic categories included normal (or nonspecific), ischemia, and scar. MPI was read nonquantitatively on film, while the E was interpreted after reviewing the digital cine loop. The intra- and inter-observer variability is displayed below using the range of Kappa values and the percent concordance in parentheses:

	Intra-observer		Inter-observer	
	MPI	E	MPI	E
Patient				
CAD	0.74-1.0 (95)	0.69–1.0 (94)	0.74–0.83 (92)	0.63–0.78 (87)
Ischemia	0.58–0.78 (86)	0.47–0.53 (92)	0.48–0.78 (85)	0.23–0.53 (90)
Regions				
CAD	0.76 (88)	0.72–0.77 (92)	0.54–0.66 (80)	0.55–0.78 (88)
Ischemia	0.65–0.68 (84)	0.23–0.49 (96)	0.42–0.53 (75)	0.02–0.27 (95)

CAD and ischemia were diagnosed by MPI in 76% and 70% and by E in 26% and 11%, respectively. Using an angiographic stenosis of > 50%, 24 of 32 pts had CAD. MPI and E were 84% and 38% sensitive, 50% and 88% specific, and had an accuracy of 76% and 50% respectively. In conclusion, acceptable interpretative consistency was noted for E and MPI. The disparity between Kappa values and concordance likely reflects differences in distribution of normal and abnormal interpretations between MPI and E.